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NEWS 4 Feb 16 TOXLINE no longer being updated
NEWS 5 Apr 03 Search Derwent WPINDEX by chemical structure
NEWS 6 Apr 24 PRF=1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
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=> s antibiotic or interferon gamma or IFN gamma or gamma interferon or gamma IFN
L1 [illegible] ANTIBIOTIC OR INTERFERON GAMMA OR IFN GAMMA OR GAMMA INTERFERON OR GAMMA IFN

=> s antibiotic or TNF or TNF alpha or tumor necrosis factor alpha or TNF beta or tumor necrosis factor beta
0 FILES SEARCHED...
L2 [illegible] ANTIBIOTIC OR TNF OR TNF ALPHA OR TUMOR NECROSIS FACTOR ALPHA OR TNF BETA OR TUMOR NECROSIS FACTOR BETA

=> s antibiotic or TNF alpha or tumor necrosis factor alpha
L3 [illegible] ANTIBIOTIC OR TNF ALPHA OR TUMOR NECROSIS FACTOR ALPHA

=> s [illegible]
L4 [illegible] ANTIBIOTIC

=> s [illegible]
L5 [illegible] ANTIBIOTIC

=> s [illegible]
L6 [illegible] ANTIBIOTIC

=> s [illegible]
L7 [illegible]

=> s [illegible]
L8 [illegible]

L4 [illegible] L3 [illegible]

=> DUP REM L4
PROCESSING COMPLETED FOR L4
L17 [illegible] DUP REM L4 [illegible] DUPLICATES REMOVED

=> DUP REM L9
PROCESSING COMPLETED FOR L9
L18 [illegible] DUP REM L9 [illegible] DUPLICATES REMOVED

=> s [illegible] AND [illegible] OR [illegible] OR [illegible]
L19 [illegible] AND [illegible] OR [illegible] OR [illegible]

=> s [illegible] AND [illegible] OR [illegible] OR [illegible]
L20 [illegible] AND [illegible] OR [illegible] OR [illegible]

=> dis 1-2 ti ab au

L17 ANSWER 1 OF 2 MEDLINE
ACCESSION NUMBER: [illegible] MEDLINE
DOCUMENT NUMBER: [illegible] PubMed ID: [illegible]
TITLE: [illegible]
[illegible]
[illegible]
AUTHOR: [illegible]
CORPORATE SOURCE: [illegible]
[illegible]
[illegible]
SOURCE: [illegible]
[illegible]
PUB. COUNTRY: United States
Journal; Article; JOURNAL ARTICLE
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980306
Last Updated on STN: 19980203
Entered Medline: 19980603

AB Organisms belonging to the Mycobacterium avium complex (MAC) are the most common bacterial pathogens in patients with **AIDS** but factors associated with the activation of cellular defense mechanisms against this atypical mycobacterium have not been defined. Peritoneal macrophages harvested from a murine MAC infection in C57 black mice are able to kill approximately [illegible] of intracellular MAC in contrast to [illegible] killing by [illegible] human and murine macrophages in vitro. The availability of human rTNF-alpha, rIFN-gamma, and rIL-2 permitted evaluation of the role of each of these lymphokines in addition, in activating macrophages in vitro to kill MAC. Human monocyte-derived macrophages were cultured in vitro, stimulated with rIL-2, rIFN-gamma, or rTNF, and then infected with MAC (serovars 1 and 4). Mouse peritoneal macrophages were harvested, cultured in vitro, and stimulated with rIFN-gamma, rTNF, or rIL-2. Viable MAC was associated with a modest increase of intracellular killing of MAC (6.4 +/- 5%) even when utilized 24 or 48 h after macrophage infection or when **administered** for 5 consecutive days after infection (24.1 +/- 4%). Both human and murine IFN-gamma were associated with increased intracellular growth of MAC (-4.1 +/- 4% for murine and -6.5 +/- 5% for human host phages). However, intracellular killing (55.4 +/- 5%) was observed with rTNF 1 day after initiation of treatment with **IFN-gamma**. This latter effect was [illegible] by anti-**TNF antibody**, whereas rIL-2 failed to augment the intracellular killing of MAC by human macrophages. rTNF plus either rIFN-gamma or rIL-2 triggered significant increases in superoxide production, but subsequent MAC killing was no greater than with rTNF alone. Treatment of macrophages with 10 U/mL of rTNF followed by rIL-2 (200 U/mL) was associated with 60% of intracellular killing. TNF seems to be an important monokine, promoting activation of mycobactericidal mechanisms in human macrophages.

AB Organisms belonging to the Mycobacterium avium complex (MAC) are the most common bacterial pathogens in patients with **AIDS** but factors associated with the activation of cellular defense mechanisms against this atypical mycobacterium have not been defined. Peritoneal macrophages. [illegible] of intracellular killing of MAC (56.4 +/- 5%) even when utilized 24 or 48 h after macrophage infection or when **administered** for 5 consecutive days after infection (24.1 +/- 4%). Both human and murine IFN-gamma were associated with increased intracellular growth. [illegible] for human macrophages. However, intracellular killing (55.4 +/- 5%) compared with control was observed after 1 day of treatment with **IFN-gamma**. This latter effect was [illegible] by anti-**TNF antibody**, whereas IL-2 alone failed to augment the intracellular killing of MAC by human macrophages. TNF plus either rIFN-gamma or rIL-2 [illegible]

L17 ANSWER 2 OF 2 AIDS [illegible] 1 AN
ACCESSION NUMBER: [illegible] CAPLUS
DOCUMENT NUMBER: [illegible]
TITLE: Anti-IL-4 monoclonal antibody and IFN-gamma administration [illegible] protection of immune hyporesponsiveness and lymphoid [illegible] in murine **AIDS**
AUTHOR(S): Wang, [illegible] Mosmann, [illegible]
CORPORATE SOURCE: [illegible]
SOURCE: [illegible]
DOCUMENT TYPE: [illegible]
LANGUAGE: English
AB [illegible] was investigated [illegible] of administration of anti-interleukin-4 (IL-4) MAb or of rat MAb [illegible] interferon-gamma (IFN-gamma), on their [illegible] after LP-BM [illegible] infection of female C57BL/6 mice with present retrovirus-induced immunosuppression and lymphoproliferation. Splenic natural killer (NK) cell activity, T- and B-cell proliferation, and T-helper type 1 (Th1) and Th2 cytokine (IL-2, IFN-gamma, IL-4 and IL-5) and monokine (IL-1 and tumor necrosis factor-alpha, TNF-alpha) responses were measured, as they are usually altered dramatically after murine retrovirus infection. Administration of IFN-gamma and anti-IL-4 significantly prevented both virus-induced suppression of splenic NK cell activity, and splenic T- and B-cell proliferative responses. They also significantly most both virus-induced increases in Th2 (but not Th1) cytokines and [illegible] and TNF-alpha). Depletion of NK cells [illegible] IFN-gamma [illegible] by spleen cells. They [illegible]

AU [illegible]

JOURNAL: [illegible]

DOCUMENT TYPE: [illegible]

LANGUAGE: English

AB This study was designed to det. if administration of anti-interleukin-4 (IL-4) monoclonal antibody (mAb), interferon-.gamma. (IFN-.gamma.) and their combination after LP-BM5 retrovirus infection of female C57BL/6 mice would prevent retrovirus-induction of immunosuppression and cytokine dysregulation. Splenic natural killer (NK) cell activity, T- and B-cell proliferation, and T-helper type 1 (Th1) and Th2 cytokine (IL-2, IFN-.gamma., IL-4 and IL-10) and monokine (IL-6 and tumor necrosis factor-.alpha., TNF-.alpha.) secretions were monitored, as they are usually altered dramatically after murine retrovirus infection. Administration of IFN-.gamma. and anti-IL-4 significantly prevented retrovirus-induced suppression of splenic NK cell activity, and splenic T- and B-cell proliferation. They also significantly allowed retrovirus-induced elevation of Th2 cytokine (IL-4 and IL-10) release and monokine (IL-6 and TNF-.alpha.) secretion by splenocytes. They prevented the loss of Th1 cytokine (IL-2 and IFN-.gamma.) release by splenocytes, and alleviated splenomegaly and hypergammaglobulinemia, precursors of signs of development of acquired immune deficiency syndrome (**AIDS**). These findings could provide insight into the roles of immune dysregulation in **AIDS** treatment as well as the mechanisms by which retrovirus infection induces cytokine dysregulation, facilitating immune deficiencies in **AIDS**.

TI **AIDS** [illegible] virus, effect on interferon

IT Antiviral agents

RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)

anti-interleukin 4 monoclonal antibody and IFN-.gamma. retardation of immune dysfunction and cytokine dysregulation during murine **AIDS**

IT Lymphokines and Cytokines

RL: ADV (Adverse effect, including toxicity); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)

(cytokine formation-inhibiting factor, anti-interleukin 4 monoclonal antibody and IFN-.gamma. administration in murine **AIDS** effect on formation of)

IT Spleen, disease

(hyperplasia, anti-interleukin 4 monoclonal antibody and IFN-.gamma. administration in murine **AIDS** effect on)

IT Lymphokines and Cytokines

RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)

(interleukin 4, anti-interleukin 4 monoclonal antibody and IFN-.gamma. retardation of immune dysfunction and cytokine dysregulation during murine **AIDS**)

IT Lymphokines and Cytokines

RL: ADV (Adverse effect, including toxicity); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)

(interleukin 4, anti-interleukin 4 monoclonal antibody and IFN-.gamma. administration in murine **AIDS** effect on formation of)

IT Lymphokines and Cytokines

RL: ADV (Adverse effect, including toxicity); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)

(interleukin 4, anti-interleukin 4 monoclonal antibody and IFN-.gamma. administration in murine **AIDS** effect on formation of)

IT Lymphokines and Cytokines

RL: ADV (Adverse effect, including toxicity); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)

tumor necrosis factor .alpha., anti-interleukin 4 monoclonal **antibody** and IFN-**.gamma.** administration in murine **AIDS** effect on formation of

IT Interferons

RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)

.gamma., anti-interleukin 4 monoclonal antibody and IFN-.gamma. retardation of immune dysfunction and cytokine dysregulation during murine **AIDS**

IT Antivirals, Biological studies

RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)

.gamma., retardation of immune dysfunction, hypergammaglobulinemia, anti-interleukin 4 monoclonal antibody and IFN-.gamma. administration in murine **AIDS** effect

L[illegible] ANSWER [illegible] [illegible] COPYRIGHT [illegible] [illegible] DUPLICATE [illegible]
ACCESSION NUMBER: [illegible] [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Treatment of autoimmune diseases, including AIDS
INVENTOR(S): **Skurkovich, Boris; Skurkovich, Simon V.**
PATENT ASSIGNEE(S): Advanced Biotherapy Concepts, Inc., USA
SOURCE: U.S., [illegible] pp., Cont.-in-part of U.S. [illegible].
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US [illegible]	A	[illegible]	US [illegible]	[illegible]
US [illegible]	A	[illegible]	US [illegible]	[illegible]
WO [illegible]	A1	[illegible]	WO [illegible]	[illegible]

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

[illegible] A [illegible] [illegible]
[illegible] [illegible]
EP [illegible] A [illegible] EP [illegible] [illegible]
R: AL, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, MC, PT, IE, SI, LT, LV, FI, RO
[illegible] A1 [illegible] US [illegible] [illegible]

PRIORITY APPLN. INFO.: US 1993-25419 A2 19930226
US 1996-771831 A 19961223
US 1997-995730 A3 19971222
WO 1997-US24266 W 19971222

AB The present disclosure concerns the treatment of a patient with autoimmune disease, including **AIDS**, by neutralizing, removing or inhibiting different types of interferons, tumor necrosis factor, HLA class II antigens, IgE, and other pathol. factors and/or their receptors, as well as neutralizing, removing or inhibiting autoantibodies, including antibodies to target cells, CD4 cells and DNA. Treatment comprises administration of an autoimmune inhibitor, or extracorporeal exposure of the patient's fluid to an immunosorbent comprising an autoimmune inhibitor, followed by return of the treated fluid to the patient, or it comprises a combined therapy involving extracorporeal immunosorption in conjunction with the administration of an autoimmune inhibitor. Combination of a plurality of two or more components selected from anti-.alpha. interferon or receptor, anti-.beta. interferon or receptor, anti-.gamma. interferon or receptor, anti-tumor necrosis factor or receptor, and anti-interleukin 6 antibodies are disclosed.

REFERENCE COUNT: 22
REFERENCE(S):
(1) Adebar; Arthritis Rheum 1989, V32(4), P1111 MEDLINE
(2) Allen, W [illegible] 1992 [illegible]
(3) Bellanti; J Immunol 1994, V152, P1197 [illegible]
(4) Blades; Lymph Kine Cyto Res 1992, V11(5), P1 [illegible]
(5) Carpentier; Int Invest 1987, V19, P1 [illegible]
(22) [illegible] AVAILABLE IN THE RE FORMAT

IT **Treatment of autoimmune diseases, including AIDS**

AN **Skurkovich, Boris; Skurkovich, Simon V.**
AB The present disclosure concerns the treatment of a patient with autoimmune disease, including **AIDS**, by neutralizing, removing or inhibiting different types of interferons, tumor necrosis factor, HLA class II antigens, IgE, and other pathol. factors and/or their receptors, as well as neutralizing, removing or inhibiting autoantibodies, including antibodies to target cells, CD4 cells and DNA. Treatment comprises administration of an autoimmune inhibitor, or extracorporeal exposure of the patient's fluid to an immunosorbent comprising an autoimmune inhibitor, followed by return of the treated fluid to the patient, or it comprises a combined therapy involving extracorporeal immunosorption in conjunction with the administration of an autoimmune inhibitor. Combination of a plurality of two or more components selected from anti-.alpha. interferon or receptor, anti-.beta. interferon or receptor, anti-.gamma. interferon or receptor, anti-tumor necrosis factor or receptor, and anti-interleukin 6 antibodies are disclosed.

IT **AIDS** disease
Ankylosing spondylitis
Autoimmune disease
Behcet's syndrome
Crohn's disease
Cell delivery systems
Extracorporeal therapy
Insulin dependent diabetes mellitus
Lupus
Multiple sclerosis
Pathogen
Psoriasis
Rheumatoid arthritis
Sarcoidosis
Systemic lupus erythematosus
Test kits
Transplant rejection
[illegible]

L[illegible] ANSWER [illegible] [illegible] COPYRIGHT [illegible] DUPLICATE [illegible]
ACCESSION NUMBER: [illegible] [illegible]
DOCUMENT NUMBER: [illegible]
TITLE: Anti-immune globulin preparation for treatment of autoimmune diseases, including AIDS
INVENTOR(S): **Skurkovich, Boris; Skurkovich, Simon V.**
PATENT ASSIGNEE(S): Advanced Bi therapy Concepts, Inc., USA
SOURCE: PCT Int. Appl., [illegible] pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

[illegible]

PATENT NO.: [illegible]

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	A1	[illegible]	WO [illegible]	[illegible]
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US [illegible]	A	[illegible]	US [illegible]	[illegible]
AU 9[illegible]	A1	[illegible]	AU [illegible]	[illegible]
AT [illegible]	B1	[illegible]		
EP 9[illegible]	A1	19991109	EP 1997-[illegible]	19971222
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1996-771631 A 19961223
US 1996-26406P P 19960822
WO 1997-US24272 W 19971222

AB The present disclosure concerns the treatment of a patient with autoimmune disease, including **AIDS**, by neutralizing, removing or inhibiting different types of interferons, tumor necrosis factor, HLA class II antigens, IgE, and other pathol. factors and/or their receptors, as well as neutralizing, removing or inhibiting autoantibodies, including antibodies to target cells, cell cells and NA. Treatment comprises administering, to the patient, an autoimmune inhibitor, or extracorporeal exposure of the patient's blood to an immunosorbent comprising an autoimmune inhibitor, followed by return of the treated blood to the patient. In an embodiment, the treatment comprises a combined therapy involving extracorporeal immunosorption in conjunction with the administration of an autoimmune inhibitor.

TI Autoimmune inhibitors and extracorporeal immunosorption for treatment of autoimmune diseases including **AIDS**

IN **Skurkovich, Boris; Skurkovich, Simon V.**

AB The present disclosure concerns the treatment of a patient with autoimmune disease, including **AIDS**, by neutralizing, removing or inhibiting different types of interferons, tumor necrosis factor, HLA class II antigens, IgE, and other pathol. factors and/or their receptors, as well as neutralizing, removing or inhibiting autoantibodies, including antibodies to target cells, cell cells and NA. Treatment comprises administering, to the patient, an autoimmune inhibitor, or extracorporeal exposure of the patient's blood to an immunosorbent comprising an autoimmune inhibitor, followed by return of the treated blood to the patient. In an embodiment, the treatment comprises a combined therapy involving extracorporeal immunosorption in conjunction with the administration of an autoimmune inhibitor.

ST autoimmune inhibitor **AIDS** treatment extracorporeal immunosorption

IT FC receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(IgE, antibodies to; autoimmune inhibitors and extracorporeal immunosorption for treatment of autoimmune diseases including **AIDS**)

IT Class II HLA antigens
IgE
Interferon .alpha.
Interferon .alpha. receptors
Interferon .gamma.
Interferon .gamma. receptors
Interleukin 1
Interleukin 1 receptors
Tumor necrosis factor receptors
Tumor necrosis factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(antibodies to; autoimmune inhibitors and extracorporeal immunosorption for treatment of autoimmune diseases including **AIDS**)

IT **AIDS** disease
Ankylosing spondylitis
Antirheumatic drugs
Autoimmune diseases
Behcet's syndrome
Crohn's disease
Drug delivery systems
Extracorporeal circulation
Graft rejection
Insulin dependent diabetes mellitus
Lupus
Multiple sclerosis
Rheumatoid arthritis
Schizophrenia
Systemic lupus erythematosus
Transplant rejection
(autoimmune inhibitors and extracorporeal immunosorption for treatment of autoimmune diseases including **AIDS**)

IT Interferon .beta.
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(autoimmune inhibitors and extracorporeal immunosorption for treatment of autoimmune diseases including **AIDS**)

IT Monoclonal antibodies
RL: BSN (Biosynthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(autoimmune inhibitors and extracorporeal immunosorption for treatment of autoimmune diseases including **AIDS**)

IT DNA
RL: [illegible]
([illegible] **AIDS**)

IT [illegible]
RL: [illegible]
(**AIDS**)

IT [illegible]
RL: [illegible]
(**AIDS**)

AIDS
IT Antibodies
RL: BSU (Biological study, unclassified); PUR (Purification or recovery); BIOL (Biological study)
(interferon, TNF, and receptor removal with extracorporeal immunoadsorbent for treatment of autoimmune diseases, including AIDS)
AIDS
IT Class II HLA antigens
RL: BSU (Biological study, unclassified); PUR (Purification or recovery); BIOL (Biological study)
(interferon, TNF, and receptor removal with extracorporeal immunoadsorbent for treatment of autoimmune diseases, including AIDS)
AIDS

L14 ANSWER 4 OF 11 MEDLINE (COPYRIGHT (C) 1997 ACS)
ACCESSION NUMBER: 94344414 MEDLINE
DOCUMENT NUMBER: 94344414
TITLE: Regulation and characterization of the interferon-alpha
receptor in patients with acquired human immunodeficiency
virus type 1 disease.
AUTHOR: Rubinstein S.; Familletti P.C.; Pestka S.
CORPORATE SOURCE: Roche Institute of Molecular Biology, Nutley, NJ 07110.
SOURCE: Journal of Interferon Research, (1994 Jun) 14 (3) 205-11.
Journal code: 8108104. ISSN: 0197-7520.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199409
ENTRY WEEK: 19940901
LAST UPDATED: 19940901
ENTRY DATE: Entered STN: 19940901
Last Updated on STN: 19940901
Entered Medline: 19940901

AB The interferon (IFN) receptor on human peripheral blood lymphocytes (PBL) was characterized in patients with acquired immunodeficiency syndrome (AIDS) and AIDS-related complex (ARC). Scatchard analysis of binding data revealed that PBL from AIDS patients had a significantly lower number of IFN-alpha receptors (IFN-alpha R) than PBL from healthy individuals. The number of IFN-alpha R on PBL from ARC patients was intermediate between those of AIDS patients and healthy individuals. The affinity of IFN-alpha R was similar in PBL from AIDS, ARC, and healthy individuals. Cross-linking studies revealed that the molecular weight of the IFN-alpha R was also similar in PBL from AIDS, ARC, and healthy individuals. Thus, the decrease in the number of IFN-alpha R on PBL from AIDS patients was not due to a change in the molecular weight of the receptor. Pretreatment of PBL from healthy individuals with human immunodeficiency virus type 1 (HIV-1) resulted in a decrease in the number of IFN-alpha R. Pretreatment of PBL from healthy individuals with recombinant gp120 also resulted in a decrease in the number of IFN-alpha R. Pretreatment of PBL from healthy individuals with recombinant gp41 did not affect the number of IFN-alpha R. These results suggest that the decrease in the number of IFN-alpha R on PBL from AIDS patients is due to the binding of HIV-1 gp120 to the IFN-alpha R. Pretreatment of PBL from healthy individuals with recombinant IFN-alpha 2a resulted in a decrease in the number of IFN-alpha R. Pretreatment of PBL from healthy individuals with recombinant IFN-gamma did not affect the number of IFN-alpha R. These results suggest that the decrease in the number of IFN-alpha R on PBL from AIDS patients is due to the binding of IFN-alpha 2a to the IFN-alpha R. Pretreatment of PBL from healthy individuals with recombinant IFN-alpha 2a resulted in a decrease in the number of IFN-alpha R. Pretreatment of PBL from healthy individuals with recombinant IFN-gamma did not affect the number of IFN-alpha R. These results suggest that the decrease in the number of IFN-alpha R on PBL from AIDS patients is due to the binding of IFN-alpha 2a to the IFN-alpha R.

L16 ANSWER 5 OF 16 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 94254741 MEDLINE
DOCUMENT NUMBER: 94254741 PubMed ID: 7515149
TITLE: A disturbance of interferon synthesis with the hyperproduction of unusual kinds of interferon can trigger autoimmune disease and play a pathogenetic role in **AIDS**: the removal of these interferons can be therapeutic.
AUTHOR: **Skurkovich S; Skurkovich B;** Bellanti J
CORPORATE AUTHOR: Advanced Biotherapy Concepts Labs, Rockville, MD.
SOURCE: MEDICAL HYPOTHESES, (1994 Jan) 42 (1) 17-24. Refs: 41
PUB. COUNTRY: ENGLAND: United Kingdom
Journal Article; (JOURNAL ARTICLE)
General Review; REVIEW
REVIEW, TUTORIAL
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199404
ENTRY DATE: Entered STN: 19940512
Last Updated in STN: 19970203
Entered Medline: 19940414
AB To examine a possible association between plasma viremia and interferon-alpha (IFN-alpha) in patients with the acquired immunodeficiency syndrome **AIDS**, we performed IFN plasma immunoadsorption by apheresis (IFN-alpha apheresis) in four volunteers with **AIDS** who had sustained levels of endogenous plasma IFN-alpha. IFN-alpha apheresis with two plasma volume exchanges was performed daily for 5 days. Clinical signs and symptoms and hematologic, virologic, and immunologic parameters were monitored. Two subjects developed anemia from phlebotomy, and one had a catheter-associated bacteremia. The IFN-alpha apheresis was effective only in transiently removing IFN-alpha. Rebound in IFN-alpha levels usually to its baseline (pretreatment) value; HIV-1 was unchanged, but there was a trend toward decreased cytopathic plasma virus burden in vitro with the procedure. Therefore, in this HIV-1-related IFN-alpha apheresis was well tolerated, but did not significantly change the HIV-1 load. IFN-alpha showed characteristics in ELISA, western blot, and biologic assays similar to the subspecies of the natural pattern. The native, recombinant, and HIV-induced IFN-alpha's demonstrated broadly cytotoxic antiviral activities. The HIV-1 IFN-alpha eluted from the column was not acid labile. The inability to isolate an excess of plasma IFN-alpha found in some patients with **AIDS** to affect viral burden likely reflects properties of the virus or of host factors independent of IFN-alpha.

L16 ANSWER 6 OF 16 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 94254741 MEDLINE
DOCUMENT NUMBER: 94254741 PubMed ID: 7515149
TITLE: A disturbance of interferon synthesis with the hyperproduction of unusual kinds of interferon can trigger autoimmune disease and play a pathogenetic role in **AIDS**: the removal of these interferons can be therapeutic.
AUTHOR: **Skurkovich S; Skurkovich B;** Bellanti J
CORPORATE AUTHOR: Advanced Biotherapy Concepts Labs, Rockville, MD.

SOURCE: MEDICAL HYPOTHESES, [illegible] 41 [illegible]. Ref: [illegible]
Journal code: MOM; [illegible] ISSN: [illegible]
PUB. COUNTRY: ENGLAND: United Kingdom
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General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: [illegible]
ENTRY DATE: Entered STN: [illegible]
Last Updated on STN: [illegible]
Entered Medline: [illegible]

AB [illegible] of interferon synthesis with the hyperproduction of unusual kinds of interferons may be the initial step which triggers autoimmune disease through a [illegible] of pathological reactions including the [illegible] of several immunological and interferon cascades. This [illegible] may result either from a genetic predisposition or from the influence of certain viruses or viral particles [illegible] together. The administration of interferons to individuals with an underlying or latent autoimmune condition can exacerbate or trigger the disease. **AIDS** has many features similar to autoimmune disease, including the hyperproduction of aberrant interferon, a type with little or no anti-HIV activity, [illegible] HIV to allow its [illegible] replication and survival. In other words, while most viruses induce normal IFN which protects the cells against viral infection, HIV induces an abnormal, defective kind of IFN which insures viral survival. The neutralization of hyperproduced interferons by polyclonal or monoclonal antibody produced in mouse, or preferably, human hybridoma, removal via extracorporeal means, or the use of antagonists which diminish the production or biological activity of these interferons can be a therapeutic approach to the management of these chronic diseases. In addition, the extracorporeal removal of different kinds of interferons, autoantibodies, autoantigens and other substances from the organism in certain pathological conditions may be an effective and safe method of treatment for autoimmune diseases and **AIDS**.

TI [illegible] interferon synthesis with the hyperproduction of unusual kinds of interferon can trigger autoimmune disease and play a pathogenetic role in **AIDS**: the removal of these interferons can be therapeutic.

AU **Skurkovich S; Skurkovich B**; Bellanti [illegible]

AB . . . together. The administration of interferons to individuals with an underlying or latent autoimmune condition can exacerbate or trigger the disease. **AIDS** has many features similar to autoimmune disease, including the hyperproduction of aberrant interferon, a type with little or no anti-HIV. . . . from the organism in certain pathological conditions may be an effective and safe method of treatment for autoimmune diseases and **AIDS**.

L16 ANSWER [illegible] OF [illegible] BIOSIS COPYRIGHT [illegible] BIOSIS
ACCESSION NUMBER: [illegible] BIOSIS
DOCUMENT NUMBER: PREV[illegible]
TITLE: Aberrant IFN may help HIV survive and replicate: Its removal in **AIDS** patients may halt this process and help restore the immune system.
AUTHOR(S): **Skurkovich, S. V. (1); Skurkovich, B.**
CORPORATE SOURCE: (1) Adv. Biotherapy Concepts Labs, Rockville, MD USA
SOURCE: Journal of Interferon Research, (1992) Vol. 12, No. SUPPL. 1, pp. S11[illegible].
Meeting Info.: Annual Meeting of the International Society for Interferon Research, Toronto, Ontario, Canada, September 28-October 2, 1992. J INTERFERON RES
ISSN: 0197-8357.
DOCUMENT TYPE: Conference
LANGUAGE: English

TI Aberrant IFN may help HIV survive and replicate: Its removal in **AIDS** patients may halt this process and help restore the immune system.

AU **Skurkovich, S. V. (1); Skurkovich, B.**

L16 ANSWER [illegible] OF 10 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: [illegible] CAPLUS
DOCUMENT NUMBER: [illegible]
TITLE: Preparation of antibodies to interferons for treatment of **AIDS**
INVENTOR(S): **Skurkovich, Simon V.; Skurkovich, Boris**
PATENT ASSIGNEE(S): [illegible], USA
SOURCE: Jpn. Kokai Tokkyo Koho, [illegible] pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: [illegible]
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
[illegible]	A	[illegible]	[illegible]	[illegible]
AU [illegible]	B	[illegible]	AU [illegible]	[illegible]
AU [illegible]	A1	[illegible]		

PRIORITY APPLN. INFO.: [illegible]

AB Human pH-unstable .alpha.-interferon was parenterally administered to sheep, the blood was subsequently isolated, and antibodies to the interferon were obtained. The antibodies were administered i.m. or i.v. at [illegible] units [illegible] 2 or 3 times a day to patients with **AIDS**.

TI Preparation of antibodies to interferons for treatment of **AIDS**

IN **Skurkovich, Simon V.; Skurkovich, Boris**

AB Human pH-unstable .alpha.-interferon was parenterally administered to sheep, the blood was subsequently isolated, and antibodies to the interferon were obtained. The antibodies were administered i.m. or i.v. at [illegible] units [illegible] 2 or 3 times a day to patients with **AIDS**.

ST antibody interferon [illegible] **AIDS**
IT Antibodies
[illegible]
[illegible] interferons, for **AIDS** treatment
IT Interferons
[illegible]
[illegible] for **AIDS** treatment

L16 ANSWER [illegible] BIOSIS COPYRIGHT [illegible] BIOSIS
ACCESSION NUMBER: [illegible] BIOSIS
DOCUMENT NUMBER: [illegible]
TITLE: METHODS [illegible] TREATING **AIDS** AND OTHER IMMUNE

AU SKURKOVICH S; SKURKOVICH

AU SKURKOVICH S; SKURKOVICH B

AB This hypothesis is a continuation of a unifying model of the interferon (IFN) system as a cascade of sequentially interacting responses of IFNs-alpha, -beta, and -gamma involved in modulation of the immune response. We propose that every antigen is an IFNogen. The first stage of immune responsiveness is associated primarily with the production of the family of IFN-alpha. In certain immunologically mediated diseases, including the autoimmune diseases and **AIDS**, disturbances in the synthesis of IFN-alpha occur with a switch to the production of predominantly acid-labile types, which have a negative immunoregulatory effect. Moreover, disturbances of IFN synthesis in the embryo or fetus can lead to deformities. Some viruses and other biological and chemical substances manifest a pathological effect by the IFN they induce. This IFN may help sustain the viruses and other substances which induce this IFN. We think it is unsafe to give patients immunoregulators in incomplete form. Thus, there is a potential danger in giving patients recombinant forms of IFNs and interleukin 2 produced in bacteria. In certain immune disorders, we may be able to treat patients by the binding or removal of hyperproduced IFNs from the body. This may lead to the restoration of immune competence and clinical improvement.

AU **Skurkovich S; Skurkovich B**; Bo...

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